transiently transfected with 1 μg of human UCP-2 promoter/luciferase vector DNA or pGL3-Basic DNA and 0.1 μg of pRL-SV40 DNA. The procedure was performed according to the attached instruction. The culture medium was exchanged to Dulbbecco's modified Eagle's MEM (Gibco Co.) containing 5% rabbit serum (Gibco Co.), and differentiation to fat cell-like cells was induced. Then, the cells were cultured at 37°C in 5% CO_2 for 24, 36, and 72 hours. After culture, the luciferase activity was detected in each culture as described in Example 2. The measurement data were presented as relative activity to the internal standard value of pRL-SV4-derived sea pansy luciferase activity. The results are shown in Figure 8. The human UCP-2 promoter/luciferase vector-derived luciferase activity was markedly higher than that of pGL3-Basic lacking the promoter in fat cell-like cells differentiated from human MG-63 cells. Therefore, the genomic DNA of human UCP-2 gene of this invention has the promoter activity reflecting the in vivo UCP-2 gene expression system in fat cell-like cells differentiated from human MG-63 cells.

Example 4 Preparation of human UCP-2 promoter-deficient vector

The human UCP-2 promoter/luciferase vector prepared in Example 2 was digested with KpnI and MluI, and the human UCP-2 promoter-deficient vector shown in Figure 9 was prepared using Deletion Kit for Kilo-Sequence (Takara Shuzo Co.) following the protocol. The plasmid digested with KpnI and MluI was purified by phenol extraction and ethanol precipitation. Then, the precipitated DNA was treated with exonuclease III and sampled every one minute, and the reaction was terminated. The samples were treated with Mung bean

nuclease and the ends were blunted. The ends were restored by Klenow fragment, and the DNA was circularized by DNA ligase. The circularized DNAs were re-treated with MluI to linearize plasmid in which deletion did not occur. *E. coli* JM 109 competent cells (Takara Shuzo Co.) were transformed with this reaction solution. The obtained deficient clone plasmids were purified by publicly known method. The molecular weights of the deficient plasmids were confirmed by agarose gel electrophoresis, and clones were selected. The base sequences of these clones were confirmed by publicly known method.

Using these plasmids, the promoter activity was measured by the procedure described in Example 2 (Figure 10).

When the base sequence containing PPRE (base number 284 - 296) shown in Example 1 was deleted, about 70% increase in the UCP-2 promoter activity was oberved, suggesting that this sequence region has a UCP-2 promoter suppressor activity. When the base sequence containing the two C/EBP binding sites (base number 1316 - 1320 and 1364 - 1368) was deleted, about 30% of the UCP-2 promoter activity decreased, suggesting that the base sequence containing these two C/EBP binding sites has a UCP-2 promoter enhancer activity. When 290 bases were deleted from the transcriptional initiation site toward downstream, no UCP-2 promoter activity was detected. Therefore, the genomic DNA of human UCP-2 gene of this invention has a promoter activity reflecting the in vivo UCP-2 gene expression control system.

INDUSTRIAL APPLICABILITY

Since UCP-2 promoter of this invention contains the regulator sequence, it has higher activity

reflecting the in vivo UCP-2 DNA expression system in human than the promoter lacking the regulator sequence. Therefore, the UCP-2 promoter of this invention can be used as a promoter inserted in vectors for treatment of human diseases and setting drug-screening systems under conditions closer to in vivo environment in human.